Hypercalcemia beyond hyperparathyroidism: broadening the etiological perspective with insights from case series

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ABSTRACT

Background: Hypercalcemia is a common electrolyte abnormality with varied etiologies and clinical manifestations. Although primary hyperparathyroidism and malignancy account for the majority of cases, less common causes may present diagnostic challenges.

Case Presentations: We report three unusual cases of hypercalcemia secondary to disseminated tuberculosis, presumptive sarcoidosis, and acute lymphoblastic leukemia. The first case involved a 44-year-old woman presenting with fever and altered sensorium. Laboratory investigations revealed severe anemia, acute kidney injury, pancreatitis, and parathyroid (PTH) independent hypercalcemia. Cerebrospinal fluid analysis was normal, but chest imaging and lymph node biopsy confirmed disseminated tuberculosis. Following initiation of antitubercular therapy, she developed progressive elevation of transaminases (AST and ALT) with rising bilirubin levels, followed by clinical deterioration that ultimately culminated in death. The second case involved a 48-year-old woman presenting with generalized body aches and a past history of cervical cancer. Malignancy-related hypercalcemia was initially suspected. However, further evaluation established a presumptive diagnosis of sarcoidosis. Management with intravenous hydration and corticosteroids led to normalization of serum calcium levels. The third case involved a 17-year-old boy who presented with fever, abdominal pain, generalized lymphadenopathy, and hepatomegaly. Investigations demonstrated anemia, acute kidney injury, and PTH-independent hypercalcemia, while peripheral blood film was unremarkable. Subsequent bone marrow examination revealed B-cell acute lymphoblastic leukemia. Although hypercalcemia improved with intravenous fluids and denosumab, he developed severe infection and died before chemotherapy could be initiated.

Conclusion: These cases illustrate that PTH-independent hypercalcemia arises from a diverse range of conditions including granulomatous infections, autoimmune disorders, and malignancies. Also, hypercalcemia can contribute substantially to morbidity manifesting as encephalopathy, acute pancreatitis, renal dysfunction, and systemic illness. A structured diagnostic approach and timely institution of definitive therapy are crucial, given the potential for rapid clinical deterioration.

Keywords: Hypercalcemia, tuberculosis, sarcoidosis, leukemia.

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Introduction

Hypercalcemia is defined as a serum calcium level above 10.5 mg/dl. Mild to moderate hypercalcemia patients present with non-specific symptoms such as fatigue, nausea, constipation, polyuria, polydipsia, and abdominal pain. On the other hand, severe hypercalcemia (>14 mg/dl) can cause life-threatening conditions such as AV block, cardiac arrest and coma. The causes of hypercalcemia can be divided as parathyroid dependent and parathyroid independent. Parathyroid-dependent causes include hyperparathyroidism due to parathyroid adenoma, hyperplasia, carcinoma, lithium therapy, and familial hypocalciuric hypercalcemia. Parathyroid-independent causes are malignancies, granulomatous diseases, and systemic diseases causing high bone turnover like hyperthyroidism. Although most common cause of hypercalcemia is hyperparathyroidism, but it is a chronic condition, and patients are often diagnosed while evaluating for asymptomatic hypercalcemia [1]. Patients presenting with acute onset severe hypercalcemia warrant urgent diagnostic workup as it can be earliest manifestation of conditions like malignancy, infections like tuberculosis, etc. We present below three cases of parathyroid-independent hypercalcemia with unusual features.

Case 1

A 44-year-old married woman presented with the complaint of low-grade fever for 4 months and altered

Table 1. *Biochemical parameters of case 1 patient.*

Lab test	Value	Lab test	Value
Hb	4g/dl (13–16)	**AST**	80U/l (14–20)
cells/L	7.5 × 10^9/L (4–11×10^9)	**ALT**	26U/l (29–33)
Platelet	120 × 10^9 WBC (150–400)	**Total bilirubin**	0.9mg/dl (<1)
MCV	80.3 Fl (80–100)	**Serum albumin**	2.0g/dl (3.4–5.4)
Urea	82mg/dl (6–24)	**Total protein**	5.8g/dl (6.0–8)
Serum creatinine	2.3mg/dl (0.3–1.3)	**Serum amylase**	255U/l (23-100)
Serum sodium	145mEq/l (135–145)	**Serum lipase**	240U/l (<67)
Serum potassium	3.7meq/l (3.5–4.5)	**Serum calcium (corrected)**	13.4mg/dl (8.5–10.2)
Random Blood sugar	143mg/dl	**Serum phosphate**	3.6mg/dl (2.5–4.5)
HIV/HbsAg/HCV ab	All non-reactive	**PTH**	6pg/ml (14–65)
CSF total cell count	Acellular (<5 cells)	**CSF ADA**	4 IU/l (<10)
CSF protein	34mg/dl (15–60)	**CSF CBNAAT**	Negative
CSF sugar	71mg/dl (45–80)	**25 OH vitamin D**	40 ng/ml (30–50)
Ionized calcium	1.48mmol/l (1.05–1.3)		

sensorium for last 5 days. There was no history of headache, seizure, limb weakness, facial asymmetry, or recent head injury. She had no chronic illness in the past. On examination, she was vitally stable with an axillary temperature of 101.4 F. On general survey, she had pallor and lymphadenopathy. Multiple subcentrimetric lymph nodes were palpable in bilateral posterior cervical triangle. Left supraclavicular lymph node was also palpable roughly 2 × 2 cm in size. Lymph nodes were soft, matted, non-tender, and freely mobile over the underlying tissue. The neurological examination revealed that she was conscious but disoriented to time, place, and person. She was able to move all four limbs spontaneously. Deep tendon reflexes were normal and symmetrical bilaterally. Both pupils were equal in size and reactive to light. Fundus examination revealed no abnormalities. The remaining cranial nerve examination and detailed sensory assessment could not be assessed. There was no neck rigidity, and Kernig's sign was absent. Abdomen was soft, generalized tenderness was present, and bowl sounds were absent. There was no organomegaly. Rest of respiratory and cardiovascular examination was unremarkable. CSF examination was done which was normal. NCCT head was normal. CEMRI brain could not be done because of deranged kidney function tests. Routine blood investigations are given below in Table 1. Patient had deranged creatinine and severe anemia for which two units of packed RBCs were infused.

Chest X-ray showed bilateral heterogenous infiltrates. HRCT chest revealed multiple centrilobular nodules with tree in bud appearance in bilateral lung field suggestive of tuberculosis [Figure 1]. Paratracheal, hilar, and mesenteric lymphadenopathy was also seen.

Left supraclavicular lymph node biopsy revealed granulomatous lymphadenitis with highly positive acid-fast

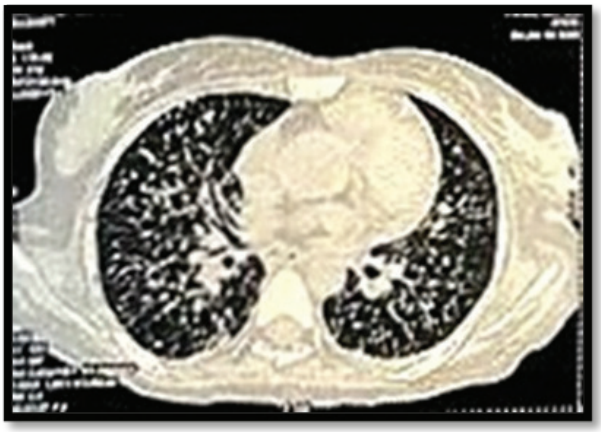

Figure 1. *HRCT showing centrilobular nodules in bilateral lung fields.*

bacilli for ZN stain using 20% H_2S0_4. It suggested disseminated tuberculosis infection-pulmonary tuberculosis with tubercular lymphadenitis. As the patient was having hypercalcemia, serum Parathyroid levels and 25 OH vitamin D levels were tested. Parathyroid levels were suppressed, and 25 OH vitamin D levels were normal. USG abdomen showed bulky pancreas. She was diagnosed with disseminated tuberculosis presenting with hypercalcemia, which was likely responsible for the development of acute pancreatitis, acute kidney injury, and hypercalcemic encephalopathy. Patient was initially managed with intravenous fluids (4L NS/day) and supportive treatment. Later once diagnosis of tuberculosis was confirmed antitubercular therapy (ATT) – Isoniazid (300 mg), rifampicin (600 mg), ethambutol (900 mg), and pyrazinamide (1500mg) daily were started. Serum calcium, her sensorium, and renal output also started improving. However, after initiation of ATT, a rise in serum AST, ALT, and bilirubin levels

Table 2. *Timeline of clinical events with corresponding trends in serum transaminases, bilirubin, calcium and creatinine levels.*

DATE	AST (U/L)	ALT (U/L)	TOTAL BILIRUBIN (mg/dl)	SERUM CALCIUM (mg/dl)	SERUM CREATININE (mg/dl)
29/5/25 (Date of admission & initiation of I/V fluids)	80	26	0.9	13.4	2.3
31/5/25	80	26	1.0	12.2	2
01/6/25 (ATT initiation)	80	25	1.0	12.2	1.9
2/6/25	82	25	1.1	12	1.6
4/6/25	600	550	3.9	10.9	1.4
6/6/25 (Modification of ATT)	960	800	10.4	10	1.4
7/6/25 (Death)	-	-	-	-	-

Table 3*. Biochemical parameters of case 2 patient.*

Lab test	Value	Lab test	Value
Hb	9.4g/dl (13–16)	**AST**	45U/l (14–20)
cells/L	7×10^9/L ($4–11 \times 10^9$)	**ALT**	23U/l (29–33)
Platelet	280×10^9 WBC (150–400)	**Total bilirubin**	0.7mg/dl (<1)
MCV	84 Fl (80–100)	**Total protein**	10g/dl (6–8)
Urea	67mg/dl (6–24)	**Serum albumin**	4.0g/dl (3.5–5.9)
Serum creatinine	3.1mg/dl (0.3–1.3)	**Albumin: globulin**	0.5
Serum sodium	136mEq/l (135–145)	**Serum calcium (corrected)**	14.3mg/dl (8.5–10.2)
Serum potassium	3.7meq/l (3.5–4.5)	**Serum phosphate**	3.7mg/dl (2.5–4.5)
Random blood sugar	116 mg/dl	**PTH**	9pg/ml (14–65)
HIV/HbsAg/HCV ab	Non-reactive	**25OH Vitamin D**	8ng/ml (30–50)
ESR	49mm/hour	**Urine complete**	Normal
Ionized calcium	1.65mmol/l (1.05–1.3)		

was observed, raising suspicion of ATT-induced hepatitis (Table 2). ATT was modified to ethambutol (900 mg) and levofloxacin (750 mg) daily. Despite management, her clinical condition deteriorated, and she subsequently died.

Case 2

A 48-year-old married woman presented with complaint of fatigue and generalized body aches for last 2 months. She had past history of cervical cancer in year 2015 diagnosed at stage 2b and managed with chemoradiotherapy. She was not on any medication currently. She had no history of fever or weight loss at present. On examination, her BP was 138/40 mmHg. She had pallor. Abdominal was soft, non-tender with no organomegaly. Respiratory, cardiovascular, and central nervous system examination was also normal. Routine lab tests were as shown in Table 3.

With past history of cervical cancer, malignancy-related hypercalcemia was suspected. CECT chest and abdomen were done which showed lymphadenopathy and consistent with WBPET CT report which revealed patchy metabolic activity in both lobes of liver, spleen, retroperitoneal and pelvic lymph nodes [Figure 2]. FDG avid subcentrimetric nodules in bilateral upper and middle lobes of lungs, enlarged right upper and lower paratracheal and bilateral hilar lymph nodes noted (garland pattern). Uterus, cervix, and bilateral adnexa showed no abnormality.

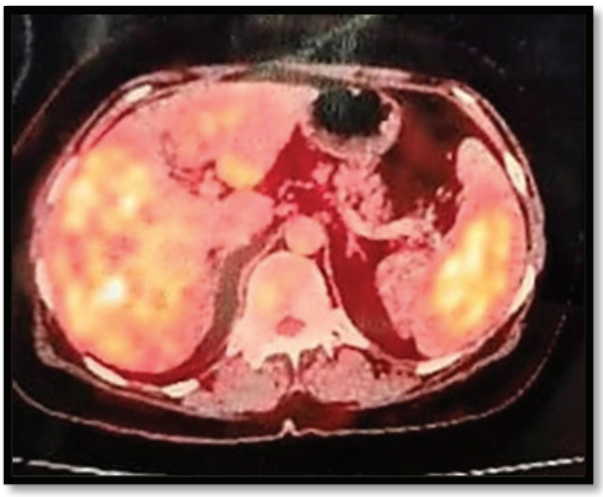

Figure 2. *PETCT showing FDG avid areas in liver, spleen and retroperitoneal lymph node.*

Serum electrophoresis was also done which did not show any M band. Endobronchial ultrasound (EBUS) guided transbronchial needle aspiration (TBNA) was performed.

Table 4. *Timeline of clinical events with corresponding trends in serum calcium and creatinine levels.*

Date	SERUM CALCIUM LEVELS (mg/dl)	SERUM CREATININE LEVELS (mg/dl)
9/7/25 (Date of admission and initiation of intravenous fluids)	14.3	3.1
10/7/25	13.8	3.0
13/7/25 (initiation of steroids)	13.9	3.0
23/7/25 (1st follow up)	12.4	2.2
6/8/25 (2nd follow up)	10.2	1.3
20/8/25 (3rd follow up, steroid tapering initiated)	10	1.2
17/9/25 (4th follow up)	9.8	1.2
20/10/25 (steroids stopped)	9.4	1.0
27/11/25	9.8	1.0
19/12/25	9.6	1.0
13/01/26	9.6	0.9

Table 5. *Biochemical parameters of the case 3 patient.*

Lab test	Value	Lab test	Value
Hb	7g/dl (13–16)	**AST**	37U/l (14–20)
cells/L	6.2 × 10^9/l (4–11×10^9)	**ALT**	30U/l (29–33)
Platelet	120 × 10^9 WBC (150–400)	**Total bilirubin**	0.9mg/dl (<1)
MCV	80.3 Fl (80–100)	**Serum albumin**	3.6g/dl (3.4–5.4)
Urea	68mg/dl (6–24)	**Total protein**	7.8g/dl (6.0–8)
Serum creatinine	3.1mg/dl (0.3–1.3)	**A:G ratio**	0.86
Serum sodium	134mEq/l (135–145)	**Serum calcium (corrected)**	19.9mg/dl (8.5–10.2)
Serum potassium	4.6meq/l (3.5–4.5)	**Serum phosphate**	5.0mg/dl (2.5–4.5)
Random Blood sugar	143mg/dl	**PTH**	9.3pg/ml (14–65)
HIV/HbsAg/HCV ab	All non-reactive	**25OH Vitamin D**	11.6ng/ml (30-50)
Serum lipase	39U/l (<67)	**Urine complete**	Normal
Serum amylase	67U/l (23–100)	ESR	72mm/hour
Ionized calcium	1.9mmol/l (1.05–1.3)		

It reported inflammatory cells, fibrocollagenous tissue, and multiple giant cells in background of few red blood cells and fibrinous material. TBNA samples were also sent for Ziehl–Neelsen (ZN) staining, cartridge-based nucleic acid amplification test (CBNAAT), acid-fast bacilli (AFB) culture, potassium hydroxide (KOH) mount, cytological examination, and cell block analysis. All of these investigations yielded negative results. Although no granuloma was seen but hypercalcemia with suppressed parathyroid and classical right paratracheal and bilateral hilar lymphadenopathy points toward sarcoidosis. Also, pattern of lung parenchymal involvement was consistent with diagnosis of sarcoidosis. Serum ACE level was on the higher side of the normal limit (44 U/l). In view of severe hypercalcemia, the patient was managed with aggressive intravenous hydration using normal saline (4–5 l/day). After a presumptive diagnosis of sarcoidosis was established, oral prednisolone (60 mg/day) was initiated. Subsequently, serum calcium levels gradually returned to normal over a month and steroids were tapered and discontinued. Also, serum creatinine improved indicating deranged kidney functions were the part of acute kidney injury from raised serum calcium as shown in Table 4.

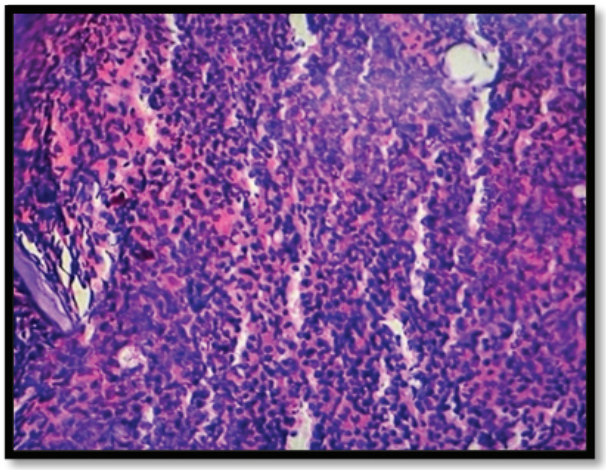

Figure 3. *Bone marrow biopsy showing near total replacement of marrow cells by lymphoblasts.*

Table 6. *Timeline of clinical events with corresponding trends in serum calcium, phosphate, magnesium and creatinine levels.*

DATE	SERUM CALCIUM (mg/dl)	SERUM PHOSPHATE (mg/dl)	SERUM CREATININE (mg/dl)	Serum magnesium (mg/dl)
10/3/26 (Date of admission and I.V fluids started)	19.9	5.0	3.1	1.8
11/3/26 (Denosumab 1st dose)	19.0	5.0	3	
13/3/26 (B cell ALL diagnosis)	17.8	5.0	2.9	1.8
15/3/26 (Urine C/S: E.Coli)	15	4.8	2.2	
17/3/26 (Denosumab second dose)	13	4.8	2.2	
19/3/26 (Pneumonitis developed)	10.3	4.8	2.2	
21/3/26	10	4.4	2.9	
22/3/26 (Death)	-	-	-	-

Case 3

A 17-year-old boy presented with complaint of low-grade fever and diffuse abdominal pain for the last 15 days. There was no history of altered bowl habits and dysuria. He had history of renal stones in the past. On examination, pallor and generalized lymphadenopathy was seen. Multiple cervical, axillary, and inguinal lymph nodes were palpable bilaterally, which were soft, rubbery, mobile with largest measuring 1.4 × 1cm. Per abdomen examination showed hepatomegaly with a liver span of 16 cm. Respiratory, cardiovascular, and central nervous system examination was within normal limits. Routine blood investigations revealed anemia, deranged renal function test, and hypercalcemia as shown in Table 5. Peripheral blood film was unremarkable.

USG abdomen showed enlarged liver with 16.5 cm size. In view of persistent abdominal pain and past history of renal stone disease NCCT abdomen was done. Although no obvious abnormality was seen in KUB region, but multiple lytic lesions were observed in lumbar vertebrae. Serum electrophoresis was done, which did not show M band. In view of persistent B symptoms, unexplained anemia and lymphadenopathy bone marrow biopsy were done, which revealed near-total replacement of marrow cells by lymphoblasts (about 90% blast cells) [Figure 3]. Immunophenotyping revealed lymphoblasts positive for CD 45, CD 19, CD 20, HLA DR, CD 13, CD 33, and Tdt. A diagnosis of B-cell acute lymphoblastic leukemia with aberrant myeloid marker expression presenting with hypercalcemia was established. Due to cost constraints, PTHrP levels were not measured. Severe hypercalcemia in the patient was managed with intravenous fluids (5–6L NS/day) and denosumab (120 mg s/c given two doses 7 days apart). Bisphosphonates were avoided due to deranged renal function tests. Blood and urine cultures were sent prior to initiate chemotherapy, which revealed significant *E. coli* growth in urine culture and chemotherapy was deferred. Appropriate antibiotics were initiated, but patient soon develop pneumonitis and ultimately died despite supportive management. (Table 6)

Discussion

Hypercalcemia has numerous etiologies. Before starting the elaborated workup, one must establish that there is true hypercalcemia and not the result of inadvertent hemoconcentration during sample collection or elevation of serum proteins such as albumin. A serial measurement of serum calcium can help in this regard. With confirmation of true hypercalcemia, a definitive diagnosis must be established as this could be the first clue to underlying disease. Immunometric assay to measure parathyroid hormone is the next crucial step. Most cases are caused by hyperparathyroidism and malignancy.

The first case represented disseminated tuberculosis-associated hypercalcemia, which was likely responsible for the development of acute pancreatitis, acute kidney injury, and hypercalcemic encephalopathy. The prevalence of hypercalcemia in tuberculosis varies from 2.3% to 28% across studies depending on cohort's dietary calcium and vitamin D levels [2]. It is seen in active tuberculosis and often reported in disseminated forms. Increased synthesis of 1,25-dihydroxyvitamin D by alveolar macrophages and CD8 T lymphocytes within granulomas is the primary mechanism leading to systemic spillover and subsequent hypercalcemia, although other mechanisms that are proposed in literature are augmented bone resorption by factors from chronically activated leukocyte in tuberculosis or isoniazid-induced conditions [3]. Few cases have been already reported in past on tuberculosis-associated hypercalcemia [4,5]. Hypercalcemia can cause varied central nervous symptoms ranging from mild fatigue to severe confusion, and stupor and coma caused by reduced neural excitability and neurotransmitter disturbances. The uniqueness of this case lies in the unusual presentation of a patient with altered sensorium, initially suggestive of meningoencephalitis. However, further evaluation revealed that the neurological manifestations were likely attributable to hypercalcemic encephalopathy secondary to disseminated tuberculosis. An individualized diagnostic approach based on clinical scenario is required

as the standard approaches and imaging might not be possible in all patients.

In the second case, hypercalcemia was presumed to be caused by sarcoidosis. Although among the granulomatous causes of hypercalcemia, sarcoidosis is the most common, but it is seen in only 10%–20% cases of sarcoidosis with a predominance in African American men [6]. Sarcoidosis-associated hypercalcemia is more common than tuberculosis-associated hypercalcemia. Here also mechanism of hypercalcemia is increased production of 1,25 dihydroxy vitamin D in the granuloma. Although few cases of sarcoidosis associated with parathyroid-related peptide leading to hypercalcemia have been reported, but sufficient evidences are still lacking [7]. Establishing the diagnosis of sarcoidosis is difficult especially when hypercalcemia is the first presentation. This is because 1,25 dihydroxy vitamin D levels may be normal with low 25 dihydroxy vitamin D. Also, ACE levels can be non-specifically elevated in parathyroid carcinoma also [8]. So, tissue diagnosis becomes indispensable. Hypercalcemia in sarcoidosis is usually mild and asymptomatic. In our patient, it was severe with previous history of cervical cancer, which made the diagnosis challenging. It highlights that hypercalcemia should not be directly attributed to malignancy without sufficient evidences even with a past history of malignancy.

The third case represented hypercalcemia of malignancy. Malignancy-related hypercalcemia has been reported in up to 20% of cancer patients [1]. It is commonly described in solid tumors such as lung cancer and renal cancer but rarely been observed in acute leukemias (<1%) [9]. The pathogenesis of hypercalcemia in hematological malignancies is attributed to two principal mechanisms in the literature: direct bone invasion by malignant cells and humoral hypercalcemia of malignancy mediated by factors such as PTHrP, TNF-α, IL-1, and IL-6 [10]. In lymphomas, an additional mechanism has been described, wherein increased production of 1,25-dihydroxyvitamin D by malignant lymphocytes and adjacent macrophages. The diagnosis of ALL in our case was challenging, as no blasts were identified on the peripheral blood smear, there was no leucocytosis, and ALL is more commonly seen in paediatric populations. Only a limited number of cases of adult B-cell ALL presenting with hypercalcemia have been reported in the literature [11,12]. We emphasize that a suspicion of ALL should be considered in background of B symptoms while evaluating hypercalcemia even if white blood cell counts are normal.

Although a limitation of our study is the lack of assessment of serum 1,25-dihydroxyvitamin D levels in patients with sarcoidosis and tuberculosis due to unavailability of the assay in our institution, the clinical features and biochemical profiles were nonetheless consistent with and supportive of the respective diagnoses. Also, serum 1,25-dihydroxyvitamin D levels need not to be always elevated in these cases.

Conclusion

Hypercalcemia was a key finding in all the patients, though with different underlying etiologies reflecting consideration of broad differential diagnosis during evaluation. PTH-independent hypercalcemia should prompt consideration of granulomatous disease and hematological malignancy. Both tuberculosis and sarcoidosis are important but often under recognized causes of hypercalcemia. Also, rarely hematological malignancies like ALL can present with hypercalcemia even before white blood cell count begin to rise or blasts become visible in peripheral blood film. These cases also highlight real-world diagnostic challenges in resource-limited settings, where adherence to standard protocols is often constrained by cost limitations and restricted access to advanced diagnostic assays.

List of abbreviations

PTH	Parathyroid
AST	Aspartate Aminotransferase
ALT	Alanine Aminotransferase
CSF	Cerebrospinal Fluid
NCCT	Non Contrast Computed Tomography
CEMRI	Contrast Enhanced Magnetic Resonance Imaging
AV block	Atrioventricular block
ACE	Angiotensin Converting enzyme

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Nil.

Conflict of interest

The authors declare that they have no conflict of interest regarding the publication of this article.

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Declaration of patient consent

The authors declare that written, informed consent was taken after discussion with the patient and family members for possibility of publication of the case along with clinical data, accompanying images and investigations. The patient and their family have been assured of maintaining anonymity during the process.

Ethical Approval

According to the policy of **Pt. B. D. Sharma Post Graduate Institute of Medical Sciences (PGIMS), Rohtak**, case report and case series study did not require approval from the Institutional Ethics Committee.

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CASE SUMMARY

PATIENT	CASE 1 44 YR FEMALE	CASE 2 48 YR FEMALE	CASE 3 17 YR MALE
Medical history	-	Cervical cancer for which patient was treated in past (2015)	-
Symptoms	Fever with altered sensorium for last 5 days	Fatigue, body aches for last few months	Fever with pain abdomen for last 15 days
Examination findings	Febrile, pallor, disoriented, absent bowl sounds, bilateral cervical, and left supraclavicular lymphadenopathy,	Normal	Pallor, generalized lymphadenopathy, hepatomegaly
Lab findings	Anemia, AKI, hypercalcemia,	Anemia, AKI, Hypercalcemia	Anemia, AKI, hypercalcemia
Diagnostic workup	HRCT chest showed pulmonary tuberculosis. Lymph node biopsy showed acid fast bacilli and granulomas	WBPET CT revealed garland pattern of hilar and right paratracheal lymph node involvement. EBUS-guided aspiration showed giant cells and inflammatory cells exclusion of other diagnosis	Bone marrow biopsy showed 90% lymphoblasts
Diagnosis	Disseminated tuberculosis-associated hypercalcemia complicated with acute pancreatitis, acute kidney injury, and hypercalcemic encephalopathy	Case of presumptive sarcoidosis with hypercalcemia and acute kidney injury	Case of B cell ALL with hypercalcemia and acute kidney injury
Management	Normal saline, ATT	Normal saline, steroids	Normal saline, denosumab
Outcome	Death	Clinically improved and stable on follow-up	Death